

Review of Contaminant Levels: Guidelines for Clandestine Drug Lab Cleanup

Introduction

The Clandestine Drug Lab Program has asked the Office of Environmental Health Assessments to review the cleanup levels for methamphetamine, mercury, lead, and volatile organic compounds presently in the Cleanup Guidelines. This review is focused on determining potential health effects associated with reoccupation of a site that has been used to produce illicit drugs, specifically methamphetamine. It is not intended to address acute exposure issues and health effects associated with first responders such as law enforcement officers and fire/HAZMAT teams, or cleanup contractors. The goal is to develop reoccupation standards that will protect the occupants (mainly children) from residual chemicals left from the production of illicit drugs.

Background

Methamphetamine is an illegal, powerfully addictive central nervous system stimulant that is easily manufactured. It is made with relatively inexpensive over the counter ingredients. Methamphetamine, commonly known as “speed,” “meth,” or “chalk,” is called “ice,” “crystal,” “crank,” or “glass” in the forms that are smoked. It is a white, odorless, colorless, bitter tasting crystalline powder that is easily dissolved for intravenous use. Currently, methamphetamine (meth) is available by physician prescription only for the treatment of narcolepsy in adults, (amphetamines, in various formulations, are used for treatment of attention deficit disorder and obesity). Because of its ease of manufacture, however, much of methamphetamine available on the street is illicitly produced in clandestine laboratories. Directions for methods of manufacture are readily available on the Internet, easily accessible and constantly changing. Over the past several years, the number of illicit labs found by drug enforcement officials has increased dramatically. Laboratories have been found in household kitchens and bathrooms, vehicles, garages, hotels, apartments, and other buildings. Many of the chemical agents used in production are caustic, corrosive, or create noxious and harmful fumes. The clandestine lab product often contains impurities resulting from incomplete reactions and inadequate purification of intermediates in final synthetic products. The reagents, contaminants, and by-products of meth manufacture place the community and health/environmental agencies charged with their cleanup at risk. Although local building officials often condemn such properties after seizure, community and health officials often seek assistance in determining the safety of these sites for future occupants.

Currently, little is known about the potential long-term health risks associated with low-level exposure to methamphetamine or precursor chemicals found at clandestine labs. High acute exposures to chemical reagents, chemical precursors, and illicit drugs have been implicated in lasting disabilities among law enforcement officers, as reported in law enforcement disability reports.

A literature search indicated that no epidemiological studies have been conducted on law enforcement personnel, operators of clandestine labs, or adults or children whom may have been exposed to the various chemicals at a lab. Low-level exposures to many chemicals have not been well studied. This, coupled with the lack of toxicity data on low-level long-term exposure to the myriad of known and unknown chemicals found at drug labs, leaves scientists to make educated guesses about potential acute and chronic health effects.

Concern is warranted because many of the chemicals whose effects have been studied at low-levels that are associated with drug labs, have been shown to produce a variety of adverse health effects, including cancer, neurological, developmental, and reproductive toxicity in animals. Mixtures have not been well studied toxicologically. Toxicologists generally assume that the combined effects of exposures to chemicals are, at least, additive. Individuals exposed at drug labs are always exposed to complex mixtures of toxic chemicals, so that it is important to note that exposure to some combinations of chemicals may produce different effects from those produced by each chemical separately.

Effects of Methamphetamine

Methamphetamine has many short and long-term effects, including effects on the heart and circulatory system, the brain, and the smooth muscles of the digestive tract, lung, bladder, and eye. Its most intense activity is on the central nervous system. It enhances the release and blocks the reuptake of the normal adrenergic neurotransmitters, the catecholamines, and may also have some direct effects on catecholamine receptors. Several studies investigating effects of methamphetamine have linked them to the neurotransmitter dopamine and at high doses to the release of serotonin. Immediate effects of this action on neurotransmitters result, according to Goodman and Gilman's text of pharmacology, in wakefulness, alertness, decreased sense of fatigue, elevation of mood, with increased ability, initiative, self-confidence and ability to concentrate; elation and euphoria, and increase in motor and speech activity. Long-term exposure leads to anorexia (loss of appetite), fatigue, increased excitability and loss of control of anger, and psychosis. The anorexic effects, alerting effects and some locomotor stimulating effects are related to increased secretion of norepinephrine at the locus ceruleus of the brain.

An increase in central dopamine mediates stereotypical behavior and some of the locomotor activities. Alteration of perception and psychotic behaviors are linked to the effects of dopamine and serotonin at the mesolimbic system. Dopamine regulates movement, emotion, motivation, and feelings of pleasure. Serotonin regulates mood, personality, affect, appetite, motor function, temperature regulation, sexual activity, sleep induction, and other basic functions. The amphetamines have a sympathomimetic effect, imitating the effects of adrenaline and noradrenaline. They cause an increase in blood pressure, heart rate, bronchial dilation, dilation of blood vessels to the skin, dilation of pupils, increased alertness, and loss of appetite. At the cellular level, they are known to interact presynaptically with the dopamine terminals, indirectly facilitating dopamine activity involving the D1 receptors. This is the basis for the psychotic action of the drug. More than 20 years of animal research shows that high doses of meth damages neuron cell-endings which do not die, but nerve ending terminals are cut back with limited regrowth.

Hazards to Infants, Children, and Pregnant Women

Health impacts on infants and young children raised in areas that were formerly used as clandestine labs, are of particular concern. Children are susceptible to clinical hazards due to their physiologic status (rapid growth, incomplete development, and rapid metabolism requiring more air and water per body weight than adults) and behaviors (crawling, hand to mouth activity, gnawing on furniture, window sills, toys). However, specific risks to infants and children associated with chronic low-level exposure to methamphetamine encountered by infants or children in a drug lab site have not been studied. Methamphetamine itself is known to cause teratogenic effects (developmental malformations), and effects on various functional body systems, (many of which subside after elimination of the drug), and behavioral effects in infants and newborns.

Additionally, the cooking of these chemicals produces vapors which permeate the interior materials of building, including sheet rock, carpets, and other porous surfaces. These chemical residues continue to volatilize from these reservoirs long after the laboratory is dismantled. This creates a potential for long-term exposure resulting in adverse health effects if a building is reoccupied without decontamination. Many of the chemicals used in methamphetamine manufacture are known to be carcinogenic, mutagenic, and teratogenic in animal and human studies.

Research on Methamphetamine and Highly Susceptible Individuals

Research into the health effects of methamphetamine and other amphetamine related drugs (e.g. ecstasy [MDMA] and MDA) has focused primarily on prenatal exposure during pregnancy in humans or high dose studies in animal. Neither of these types of studies were designed to answer the question “are there potential health effects from exposure to chronic, low levels of methamphetamine to infants, toddlers, and children.” These studies do, however, allow us to understand the mechanism with which these toxic substances act and from this, apply this information in estimating the potential risks to children.

Human Studies

The incidence of infants born with evidence of illicit drug exposure greatly increased over the past two decades (Dixon, 1989). Considerable research has been devoted to determining the adverse effects upon the infant when the mother uses methamphetamine during pregnancy. The course of withdrawal and outcome for heroin- and methadone-exposed children has been well described. Signs of neonatal narcotic withdrawal are quantified in scoring formats such as that developed by Finnegan (Dixon, 1989). A Swedish study which evaluated the effects of prenatal exposure to amphetamines showed an increase in preterm labor, placental abruption, fetal distress, and postpartum hemorrhage. Infants that were exposed were smaller and had feeding difficulties and their development was described as “very slow” (Ericson et. Al, 1978). Similarly, Oro and Dixon (1987) showed that infants born to mothers who used methamphetamine during pregnancy had altered neonatal behavioral patterns (abnormal sleep patterns, poor feeding, tremors, and hypertonia) as demonstrated by the Finnegan withdrawal scoring scheme.

Visual recognition memory testing was used to evaluate cognition in infants exposed prenatally to amphetamines. Study findings showed that prenatal exposure resulted in significantly lower intelligence testing scores compared to nonexposed infants and that exposed infants may be at risk for later subtle neurological abnormalities (Struthers and Hansen, 1992). Numerous physical malformations resulting from prenatal exposure to amphetamine and methamphetamine have been reported. These include cleft lip, cardiac defects, low birth weight, reduced head circumference, biliary atresia, cerebral hemorrhage, low body fat, systolic murmur, and undescended testes (Plessinger, 1998). Several case reports, involving either prenatal exposure or exposure to infants, adds to the growing concern over early exposure to methamphetamine. One such case report involved a woman who had taken methamphetamine five hours prior to giving birth to twins that died a few hours after birth (Bost, 1989). Upon autopsy it was determined that the infants blood methamphetamine concentrations were 280 ng/ml. An 11-month-old infant developed transient cortical blindness after accidental exposure to an unknown quantity of methamphetamine. Blood concentration of methamphetamine was 88 ng/ml (Gospe 1995). A third case report involved the criminal conviction of a woman for killing her two-month-old infant by administering a lethal quantity of methamphetamine via breast-feeding. The methamphetamine blood level of the infant at autopsy was 39 ng/ml, comparable with levels seen in narcolepsy therapy of adults (Ariagno et. al, 1995). It should be noted that other, unmeasured contaminants, may have contributed to the effects seen in these case reports.

Animal Studies

Numerous animal studies have been conducted to determine the effect of methamphetamine exposure in adults as well as effects during pregnancy. Compelling evidence exists demonstrating the adverse effects of meth on the central nervous system of rats and monkeys. For instance, selective long-lasting reductions in the level of serotonin, the number of serotonin uptake and neurochemical deficits due to serotonin nerve terminal degeneration, have been seen in rats (Ricaurte et. al, 1985). Similarly, a study conducted by Slikker et. al, (1988) showed selective reductions in brain serotonin and dopamine concentrations, along with histologically observed neuronal degeneration in both rats and monkeys suggesting destruction of serotonergic nerve terminals.

Determining a Cleanup Level that is Safe for Infants and Children

Members of the steering committee have suggested that a “safe dose” for cleanup could be derived by dividing a therapeutic dose of methamphetamine or amphetamine by a safety factor. Methamphetamine is only prescribed for narcolepsy in adults. There are no clinical reasons for prescribing methamphetamine to infants or children. Some forms of amphetamine are prescribed for Attention Deficit Disorder for children that have been diagnosed with this problem. However, such a controlled substance should only be prescribed on the basis of legitimate therapeutic need and not as a blanket exposure level. Since the effects of methamphetamine on infants and children at low levels of exposure are not well understood (in contrast to those of the pharmaceutical levels of amphetamine), listing an allowable level of exposure based on pharmaceutical effects of amphetamine in children gives the impression of “prescribing” an illegal substance.

Aside from Department of Health not being legally empowered to prescribe a controlled substance, the severity of effect, the lack of data on developmental effects thresholds, and the occurrence of effects at therapeutic adult levels, make it unfeasible from a public health standpoint, to set guidance by dividing a therapeutic dose of amphetamine by a safety factor, in order to arrive at an acceptable exposure to methamphetamine. Dermal uptake of methamphetamine by children from contact with contaminated surfaces has not been studied.

In the WAC (WAC 246-205-010), decontamination is defined as “the process of reducing levels of known contaminants to the lowest practical level using current available methods and processes.” Ideally, no exposure is preferred, but since it is not possible to define cleanup levels below the detection **limit ($>0.002 \text{ ug/ft}^2$)**, the value **5 ug/ft^2** , was chosen as an interim achievable standard of cleanliness. This was seen as a compromise that would give a measurable value unlikely to cause harm to children crawling around.

The 5 ug/ft^2 is not a “safe level” standard, since no one knows how much an infant or child would absorb via skin, or get as an oral dose from putting hands that have been in contact with the contaminant in its mouth. It is somewhat unlikely that a concentration approaching the effective doses, or even one or two orders of magnitude lower, would be absorbed in a short enough period of time. However, methods for detecting methamphetamine in samples have improved since the interim level of 5 ug/ft^2 was agreed upon. A readily achievable value of 0.1 ug/100 cm^2 (note change in units) is now possible. We recommend that this value be used in the rule to serve as a protective level for infants and toddlers who are most likely to be exposed through contact with contaminated surfaces.

Lead and Mercury

Currently, much more is known about the chronic low-level exposure to lead and mercury in infants and children. Lead and mercury are especially toxic for infants and toddlers. Young children crawling and playing on carpet permeated with heavy metals are potential victims for permanent brain damage and other neurological damage. We recommend that current cleanup levels for lead and mercury should be put forward in the rule. However, testing and cleanup for lead and mercury should only be required if evidence exists that these precursors were used in methamphetamine production, or if there is evidence of the amalgam method of production being employed.

Volatile Organic Compounds

We recommend that current levels for cleanup of VOCs be put forward in the rule.

Summary of Recommendations

- Methamphetamine cleanup standard be set at 0.1 ug/100 cm^2 (one hundred square centimeters wipe sample).
- Mercury and lead cleanup standards stay at current levels (**50 ng/m^3 in air** and **20 ug/ft^2** for a one square foot wipe sample, respectively).

- VOCs cleanup standard be less than **1 ppm total hydrocarbons** threshold limit value for an eight-hour time weighted average (TLV).

References

Ariagno R, Karch SB, Middleberg R, Stephens BG, Valdes-Dapena M. Methamphetamine ingestion by breast-feeding mother and her infant's death: People v Henderson. JAMA. 1995;274(3):215.

Bost RO. Tissue distribution of methamphetamine and amphetamine in premature infants. J Anal Tox. 1989;13:300-302.

Dixon SD. Effects of transplacental exposure to cocaine and methamphetamine on the neonate. West J Med. 1989;150:436-442.

Ericksson M, Larsson C, and Windbladh B. The influence of amphetamine addiction on pregnancy and the newborn infant. Acta Paediatr Scand. 1978;67:95-99.

Goodman and Gillman's The Pharmacological Basis of Therapeutics. 8th edition. Gilman, A.G., Rall, T.W., Nies, A.S., Taylor, P., editors Pergamon Press, New York. 1990.

Gospe SM. Transient cortical blindness in an infant exposed to methamphetamine. Ann Emerg Med. 1995;26:380-382.

Oro AS and Dixon SD. Prenatal cocaine and methamphetamine exposure: maternal and neonatal correlates. J Pediatr. 1987;111:571-578.

Plessinger MA. Prenatal exposure to amphetamines. Obs Gyn Clin North Am. 1998;25(1):119-138.

Ricaurte G, Bryan G, Strauss L, Seiden L, and Schuster C. Hallucinogenic amphetamine selectively destroys brain serotonin nerve terminals. Science. 1985;229:986-988.

Slikker W, Ali SF, Scallet AC, Frith CH, Newport GD, and Bailey JR. Neurochemical and neurohistological alterations in the rat and monkey produced by orally administered methylenedioxymethamphetamine (MDMA). Tox Appl Pharm. 1988;94:448-457.

Struthers MD and Hansen RL. Visual recognition memory in drug-exposed infants. J Dev Behav Pediatr. 1992;13(2):108-111.